to heal soundly—all those at least which are going to heal.

Month by month my vagotomy cases improve. The primary or neurogenic phase of secretion is abolished and with it the symptoms, but there is nothing to substantiate the belief that the secretory function of the hormonal phase of secretion is grossly disturbed. Once these post-vagotomy cases become stabilized they seem to be perfectly comfortable, they may have more "gas" than the gastrectomies, but there is a long period when they are in minor distress from one or other cause.

This time vagotomy is here to stay. Earlier in the century, attempts were made to divide the nerves to the ulcer. Now we are dividing vagi to the stomach which contains the ulcer, confident that the ulcer itself is probably just the local manifestation of a more general and ill-understood phenomenon. More and more we are treating patients who have peptic ulcer, less and less we are treating ulcer itself. The importance of hypoproteinæmic states on the maintenance of ulcer diathesis is becoming more clearly understood. The ramifications of vagotomy are wide and reports are filtering through of highly satisfactory results following its use in ulcerative colitis and kindred disorders. It has stimulated a tremendous interest in the physiology and chemistry of gastric secretion, it has already resulted in serious questionings as to the adequacy of our methods of testing and measuring gastric secretion in our clinical laboratories. It has brought us additional proof that all peptic ulcers do not heal with the development of anacidity. The old battle cry "No acid, no peptic ulcer" may still be true with respect to the genesis of ulcer, but is definitely not true with respect to its perpetuation. They can and do remain unhealed in the achlorhydric stomach.

It took me ten years to assess the value of gastro-enterostomy. It took another ten to properly evaluate gastric resection and it will probably require ten to clearly establish the position of vagotomy. From my own personal experience I believe that its greatest value is in the eradication of stubborn pockets of difficulty as in marginal ulceration and high perforating gastric ulcer. Its advantages in these situations, in my opinion, far outweigh its disadvantages.

CASE REPORTS

ACUTE PERICARDITIS SIMULATING ACUTE CORONARY OCCLUSION

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I would like to report a recent case of acute pericarditis simulating acute coronary occlusion because of its rarity and also because of the interest it aroused among our members. I was only able to find one reference to this condition. Drs. Barnes and Burchell of Mayo's Clinic[1] reported 14 cases in which 9 were definitely diagnosed as acute pericarditis.

These authorities state that there is a distinctive form of pericarditis of a benign non-suppurative nature. Pain in the chest is the most outstanding complaint. There are patients regarding whom the crucial question arises whether the diagnosis should be acute coronary occlusion or acute pericarditis. The problem is complicated still further by the well known fact that patients who have acute coronary occlusion may have pericarditis as an adjunct. The importance of making the correct diagnosis is obvious.

In some of these cases at no time are there any x-ray changes. This was found in the patient herewith reported. The correct diagnosis is made by means of the electrocardiograph. There will be upward displacement in one or a combination of the standard leads. The RT segment is either concave upward or forms a straight line from its origin in the R wave to the crest of the T wave. This is to be contrasted with the upward convexity of the elevated RT segment in coronary occlusion. Reciprocal deviations of RS—T segments on Lead 1 and 3, such as may occur after acute coronary occlusion, rarely occur in uncomplicated pericarditis. The T waves in acute pericarditis primarily tend to undergo one of two changes; they may become exaggerated in amplitude or sharply peaked as in our case, or they may be rounded with a dome shape. There is no Q or T pattern.

Within one to six weeks after the acute phase of pericarditis subsides, the electrocardiogram returns to normal or begins to approach normal limits. In the case here reported a normal

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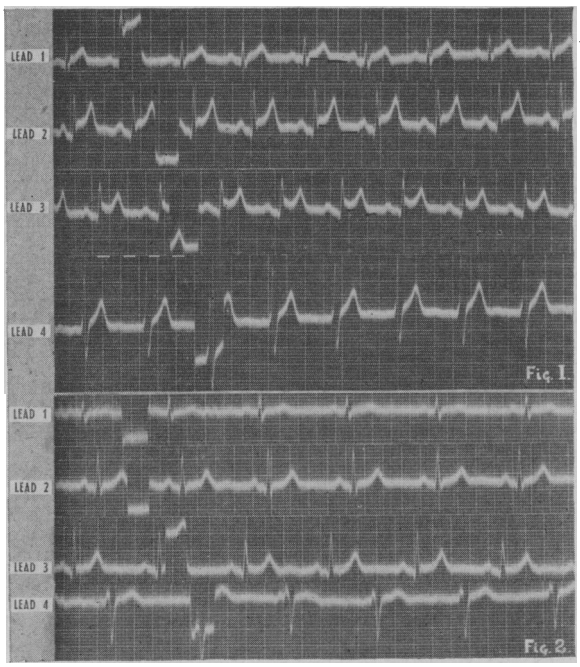
electrocardiogram was obtained in eight days. There are no traces of the Q or T pattern in the standard leads and no persistence of Q or absence of R in the precordial apical leads to suggest a previous occlusion.

Mr. M.K., aged 38, construction foreman, was admitted to Brandon General Hospital, December 7, 1947. Entrance complaints were severe pain in mid chest radiating to neck and arms. Patient was cyanotic and very dyspnœic. He was given morphine and continuous oxygen in Fowler's position. Temperature was 100.2° and pulse 100. Heart sounds and pulse were very faint. No friction rub was heard. The patient had had a cold with gradually increasing pain in the mid chest for one week prior to admission. He thought that the pain became more severe after lifting a heavy pole on December 6, 1947. This brought up the question of a possible hæmopericardium.



December 8, 1947.—X-ray of chest showed no abnormalities; white blood cells 18,550; sedimentation rate 16 mm. in one hour; urinalysis negative; temperature 99.3; pulse 92. An electrocardiogram taken December 8, 1947, (Fig. 1) showed markedly elevated ST segments in Lead 1, 2, and 3. The T waves were high and peaked. The ST curve was concave upwards. These findings were consistent with a diagnosis of acute pericarditis but in view of a negative x-ray we were hard put to concede this diagnosis over coronary occlusion. The patient showed marked improvement with bed rest, sedation and hyperoxygenation. On December 16, he was up and felt perfectly well with normal temperature, a pulse of 60, blood pressure 128/84, normal x-ray findings in the chest. An electrocardiogram then taken (Fig. 2) was completely normal. He was discharged from hospital on December 19, 1947.

When the patient was seen again on January 3, 1948, he had no symptoms. The chest x-ray was again normal. On January 7, one month after his attack, he was back at work as a construction foreman.

REFERENCE

1. BARNES, A. R. AND BURCHELL, H. B.: *Am. Heart J.*, February, 1942.

217 Tenth Street.

HÆMANGIOMA OF THE URINARY BLADDER*

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Reports of renal hæmangioma are not uncommon in the current literature but a review of the literature of the past ten years reveals a paucity of reports of angioma of the urinary bladder. Hæmangioma of the urinary bladder are of such rarity that in the textbooks of urology they are dismissed with mere mention. Macalpine, in 1930, reviewed 20 cases of hæmangioma of the urinary bladder and added two of his own. In only part of the reports was there histologic confirmation. The case presented here is deemed of sufficient interest to report.

G.G., male, aged 66, was admitted to the Urologic Service, Shaughnessy Hospital, December 12, 1946. Complaint was (1) hæmaturia, off and on for the past three months, the amounts of blood lost becoming progressively greater. On day of admission there was a large hæmorrhage with blood clots and the patient went into retention. (2) Frequency and nocturia, days every three hours, nights every one and one-half hours for the past several years, accompanied by (3) small stream and some delay in starting stream. These symptoms have become worse during the past 2 months. (4) Dysuria for the past 4 months. (5) The patient has been under medical treatment for years for a cardiac condition.

On admission he complained of severe dyspnœa on exertion. The blood pressure was 160/80; pulse 130; heart enlarged to the left. Chest emphysematous; râles at both bases. Abdomen negative, no C.V. angle tenderness, kidneys not palpable, suprapubic mass which disappeared following catheter drainage and irrigation with normal saline. Prostate slightly enlarged, grade 1, firm but glandular. Urinalysis: pH. 7.5; grossly bloody; non-protein nitrogen 40 mgm. %; Kahn negative; Hb. 50%. On continuous bladder lavage through F. 22 irrigating catheter the bleeding cleared, and then urinalysis showed pH. 7.0; white blood cells one plus; red blood cells four plus; urine culture, no growth, phenolsulphonphthalein test, 30% in 2 hours.

Excretory urogram: Upper urinary tract appears within normal limits. Moderate sized filling defect in right dome of bladder—probable neoplasm. There is a defect at base of bladder, probably due to prostatic intrusion. There is delay in excretion and at the end of one hour there is good concentration of dye present in both kidney pelves.

The patient was given two blood transfusions of 250 c.c. each. His condition had improved sufficiently that on December 19, 1946 a cystoscopy was done. This showed grade 3 intraurethral lateral intrusion with grade 2 median commissural lobe hypertrophy. There is a large, broad base, fungating tumour of the bladder, probably carcinoma, over the antero-lateral wall on the right side and obscuring the right uretheral orifice. There is an extension of the growth on to the anterior aspect of the vesical neck. Marked trabeculation present.

Bimanual examination showed no fixation of the bladder floor nor induration of either the bladder floor or the vault. There was moderately advanced prostatism.

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